in the range of historical controls. To address this issue, our laboratory has instituted the use of multiple independent controls in this short-term bioassay. Any biological response displays a range of values; hence, independent, duplicate negative controls are used to define the variability of the untreated cell population. The potent carcinogen, 7,12-dimethylbenzanthracene, is used as a positive control at two doses; the high dose defines the variability of the treated cell population,and the low dose is used to define the resolving power of the assay. Determining the variability and resolving power of the assay system, along with developing a data base from a large number of experiments, provides a statistical basis for detecting weak carcinogenic substances.
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GENOTOXICITY OF 1,3-BUTADIENE. ASSESSMENT BY THE UNSCHEDULED DNA SYNTHESIS ASSAY IN B6C3F1 MICE AND SPRAGUE-DAWLEY RATS IN VIVO AND IN VITRO. D. R. Vincent*, G. Theall Arce and A. M. Sarrif. (Introd. by D. A. Vlachos) Haskell Laboratory, E. I. du Pont de Nemours and Co., Inc., Elkton Rd., Newark, DE 19714.

1,3-Butadiene (BD) has been shown to cause a variety of tumors in mice, but induces few tumors in rats. BD was tested in the in vivo/in vitro UDS assay, and two BD metabolites, butadiene monoxide (BMO) and butadiene diepoxide (BDE), were tested in the in vitro UDS assay. Male B6C3F1 mice and Sprague-Dawley rats were used in these experiments. Two exposure/sampling protocols were employed in the in vivo/in vitro experiment: (a) 6 hours exposure on the first day, 3 hours on the second day/sampled 2 hours after the second exposure, and (b) 6 hours exposure per day for 2 days/sampled 18 hours after the second exposure. The animals were exposed nose only to either air or air containing 10,000 ppm BD. This concentration of BD is 16-times the minimum level shown to induce tumors in mice. No UDS was evident in either species at the concentration of BD tested in this assay. Test concentrations for both BMO and BDE ranged from 1 uM to 150 uM in the in vitro experiment. Both BMO and BDE were cytotoxic at test concentrations above 5 uM, but neither metabolite induced UDS. Although the liver figures prominently in the metabolism of BD, this organ showed limited tumor formation in the carcinogenicity studies. These results indicate that BD does not induce DNA damage that is repaired by an excision-repair mechanism in the liver.

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A FRAMESHIFT DETECTION ASSAY IN THE YEAST *SACCHAROMYCES CEREVISIAE* WHICH DETECTS BASE-PAIR ADDITIONS AND DELETIONS AS SEPARABLE EVENTS. R. C. von Borstel, E. A. Savage*, P. O. Andersson* and U. G. G. Hennig, Department of Genetics, University of Alberta, Edmonton, Canada, T6G 2E9

A strain has been developed comprising three mutant loci in yeast which respond to the same frameshift suppressors. These mutants are *his4-38*, *leu2-3*, and *hom3-10*. The first two mutants were provided by G. Fink, and *hom3-10* was provided by G.E. Magni. Mutations can be selected at any of the three loci on synthetic complete medium minus the metabolite for the mutant being reverted. The reverted colonies are replica plated to determine auxotrophic requirements. Those revertants which also revert one or both of the the other two frameshift mutants are adjudged to be frameshift suppressors, revertible by addition of one base to the anticodon region of the suppressing tRNA gene. Those that do not suppress are assumed to be -1 intragenic frameshift suppressors or -1 back mutations of the original mutant base-pair. It is possible that at least a few of these are +1 extragenic suppressors which suppress only the original frameshift mutant, but the frequency of this class has not yet been determined. Spontaneous reversion rate measurements of the *leu2-3* and *his4-38* mutants indicates that reversion on limiting leucine takes place at a substantially higher spontaneous rate (1.63×10^{-8} mutations/cell/generation) than reversion on limiting histidine (3.5×10^{-10}). The reversion rate for suppressors of *leu2-3* was 9×10^{-10}, and for suppressors of *his4-38* it was 2×10^{-11}.
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THIOL LEVELS IN RELATION TO PHOTOSENSITIZED MORTALITY IN THE HOUSE FLY AND IN E. COLI. John M. Wages, Jr.[1,2], James R. Heitz*[1], and Philip E. Hartman[2], Department of Biochemistry, Mississippi State University, Miss. State, MS 39762[1], and Department of Biology, The Johns Hopkins University, Baltimore, MD 21218[2].

Photosensitizers are common in nature and probably exist as an anti-predation agent in plants (Ames, 1983 Science 221:1256). In addition, photosensitizing halogenated

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